

## A Most Irregular Threat

### Old Gas Regulators Can Present Mercury Exposure

Residential gas regulators reduce the pressure of gas from main feeder lines to usable levels for household pipes. Gas regulators built before 1961 were commonly located within dwellings and incorporated a component that contained, on average, 136 grams

This case and a cluster of similar cases quickly caught the attention of area authorities. Ultimately, two area gas companies were required to conduct inspections in 361,000 homes where their employees or subcontractors had replaced old gas regulators. Free urine mercury screenings were offered to concerned residents.

Of the 301,000 homes inspected by one of the companies, 1,308 (0.43%) were found to be contaminated, and 1,033 were remediated. Of 60,000 homes screened by the other company, 55 (0.09%) were found to be contaminated and were remediated.

The risk was considerably higher in homes where the equipment had been replaced by one particular subcontractor—of 120 homes screened, 20 (16.7%) were found to have been contaminated by mercury.

Of the 625 residents who elected to undergo urine mercury screenings, 9 (1.4%) had positive bioassays, defined as a 24-hour urine mercury concentration equal to or higher than 10 micrograms per liter. Although none of the subjects showed overt symptoms of mercury poisoning, as the authors point out, the screenings were not designed to detect subclinical effects of mercury exposure.

Interestingly, positive urine mercury in residents was more strongly associated with elevated air mercury concentrations on the first floor of the homes than with elevated basement air levels, even though the air concentrations were considerably higher in basements, where the spills typically occurred. The authors attribute this result to the simple

fact that people generally spend less time in their basements than in the living quarters aboveground.

The Chicago episode not only revealed a previously unidentified environmental exposure hazard but also provided valuable lessons on how to respond quickly and efficiently. As the authors note, gas companies and their subcontractors, clinicians, public health and environmental officials, and residents all need to be aware of the potential for contamination in older homes or other buildings where mercury-containing gas regulators have been replaced in the past, or where they may still exist. —Ernie Hood



**Old equipment poses new problem.** Old gas regulators that contain mercury can present a potential health hazard to residents if care is not taken when they are replaced.

(about 2 teaspoons) of elemental mercury. Newer units do not contain mercury, but as a group of Chicago-area researchers report, careless replacement of the older units can result in potentially hazardous mercury contamination [*EHP* 114:848–852; Hryhorczuk et al.].

The authors describe how this newly identified source of residential mercury exposure came to light in 2000, when a suburban Chicago family discovered a pool of the silvery element on the floor of their basement workroom six weeks after a gas company contractor removed and replaced their gas regulator and meter. An investigation showed that the air in the house contained elevated levels of mercury vapor, and the father and the 9-year-old son, who spent more time in the basement area than the mother, had blood and urine mercury levels above recommended background limits. Short-term exposure to high levels of metallic mercury vapors can cause lung damage, nausea, vomiting, diarrhea, elevated blood pressure or heart rate, skin rash, and eye irritation.

Fortunately, ventilation and remediation of the home brought mercury concentrations in the home's air down to safe levels, and several weeks after exposure ceased, the father's and son's blood and urine mercury levels returned to normal background ranges. Neither ever manifested overt clinical signs of mercury poisoning.

## PCBs Are Endocrine Disruptors

### Mixture Affects Reproductive Development in Female Mice

Polychlorinated biphenyls (PCBs) are a broad group of chemicals that includes 209 aromatic chlorinated hydrocarbons used for products ranging from fluorescent light fixtures to coolant fluids

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inside parts of consumer electronics. Short-term exposure to large amounts of PCBs can cause liver damage; the effects of smaller concentrations can be more subtle, affecting the reproductive development of children of exposed mothers. But are these compounds actually endocrine disruptors? New work by researchers at the Mount Sinai School of Medicine confirms that they can be, and for the first time connects a molecular mechanism to the life-long phenotypic changes seen after exposure to environmental estrogens [*EHP* 114:898–904; Ma and Sassoon].

Sold predominantly as mixtures under the trade names Aroclor and Pyranol, PCBs have been banned in the United States since 1977 and are out of use or highly restricted throughout much of the world. These compounds, which are fat-soluble and structurally similar to DDT, are highly stable: PCBs made and used for nearly 50 years before the ban remain in the environment and are found throughout the food chain, including in human tissues and breast milk.

The researchers tested the hypothesis that PCBs are endocrine disruptors by comparing their effects on expression of *Wnt7a* in mice. Down-regulation of *Wnt7a* is a known factor in the reproductive deficits found in mice exposed to diethylstilbestrol (DES), a synthetic estrogen once used to supplement estrogen levels in pregnant women. After exposure to DES, development of the reproductive system is impaired when production of the regulatory protein coded by *Wnt7a* is temporarily squelched. A passing loss of appropriate regulation in very young animals leads to reproductive changes—for example, in the number of uterine glands or the thickness of the myometrium, the uterus's muscular outer layer—that give way to more pronounced changes through life. This suggests that exposed organisms are sent down an abnormal developmental path from which they cannot return.

Using *in situ* hybridization, immunohistochemistry, and quantitative reverse-transcriptase polymerase chain reaction, the authors showed that the effects of environmentally relevant levels of Aroclor 1254, a commercial mix of PCBs, were similar but not identical to those of low levels of DES. Both led to decreases in expression of the *Wnt7a* regulatory gene and showed qualitative changes in uterine development like those described above. The authors also demonstrated that measurement of *Wnt7a* provides a molecular tool that lays to rest any doubt that PCBs are endocrine disruptors.

The authors further showed that genetic makeup matters in terms of vulnerability to endocrine disruption. Mice with only one good copy of the *Wnt7a* gene were more sensitive to DES or PCBs than mice with both copies intact, providing a concrete example of how gene variation can leave some individuals more susceptible than others to the effects of PCB exposure. —Victoria McGovern

## Putting a Load on Your Bones

### Low-Level Cadmium Exposure and Osteoporosis

High-level exposure to cadmium is known to cause bone damage, including osteoporosis, but the effects of low-level exposure have been less clear. A group of Swedish researchers now shows for the first time that low-level exposure to cadmium also can be associated with negative effects on bone in humans [*EHP* 114:830–834; Åkesson et al.]. Although the impact may be slight, even a limited role for cadmium in the etiology of osteoporosis could be important at the population level, given the prevalence of osteoporosis and our ubiquitous, life-long low-level exposure to the substance through diet.

Unlike lead (another contributor to osteoporosis), which is retained in bone tissue, cadmium is retained mainly in the kidneys. Exposure mostly comes from cereals, vegetables, shellfish, and tobacco, all of which absorb cadmium. Some cadmium occurs naturally, and more is released in industrial emissions and vehicle exhaust.

To investigate associations between cadmium retention and bone effects, the scientists assessed a cohort of women ranging in age from 53 to 64 years. This segment of the population is the most susceptible to both cadmium retention (which appears to decrease slightly past this point) and osteoporosis. The 820 subjects were recruited from a large ( $n = 10,766$ ) population-based survey of upper-middle-aged women in the community of Lund, Sweden. The lack of known history of excessive cadmium contamination in this area implied that exposures were fairly constant over time.



**From cadmium to canes?** New data show that even low-level exposure to cadmium may contribute to osteoporosis.

The team measured cadmium in blood and urine; lead in blood; several biochemical markers of bone metabolism; and forearm bone mineral density (BMD), a test used to assess osteoporosis status. Statistical analysis of the results incorporated a comprehensive array of potential confounders and effect modifiers, including weight, menopausal status, use of hormone replacement therapy, age at menarche, alcohol consumption, smoking history, and physical activity level.

Increasing urinary cadmium concentrations were associated with decreasing BMD. Furthermore, urinary cadmium was negatively associated with parathyroid hormone (a bone metabolism hormone) and positively associated with urinary deoxypyridinoline (a bone resorption marker). Those associations were present even in the subgroup with the lowest cadmium exposure—those who had never smoked. The study also showed that the negative bone effects appeared to intensify after menopause.

The authors calculated that women in the 99th percentile of urinary cadmium concentration had an average of 5–6% lower BMD than those in the 1st percentile. This difference was similar to what could be expected from a 6-year-greater age or 11-kilogram-lower body weight. Although the researchers acknowledge that this contribution of low-level cadmium exposure to the development of osteoporosis is small, they emphasize that the observed effects should be considered “early signals of potentially more adverse health effects.” The findings thus lend increased urgency to efforts to reduce cadmium pollution of the environment. —**Ernie Hood**

## The Public Health Payoff of “No Smoking Allowed”

### Quantifying Decreases in SHS Exposure

Secondhand smoke (SHS), the cigarette smoke involuntarily inhaled by a nonsmoker, was recognized as a serious health problem as early as 1972. Studies have shown that SHS causes lung

cancer and leads to other adverse effects, including lower respiratory tract infections, bronchitis, pneumonia, fluid in the middle ear, and sudden infant death syndrome. Fortunately, a new report by researchers at the CDC shows that over the last 14 years, SHS exposure has decreased substantially—by an average of 70% in people over the age of 4, regardless of sex, age, or race [*EHP* 114:853–858; Pirkle et al.].

The researchers analyzed data from the National Health and Nutrition Examination Survey (NHANES), conducted by the CDC’s National Center for Health Statistics, which collected data on the U.S. population during four distinct time periods from 1988 through 2002. Over the 14-year span, 29,849 nonsmoking participants completed a home interview followed by a physical exam.

SHS exposure was measured by testing blood samples from each participant for serum levels of cotinine, the primary by-product formed when the body metabolizes nicotine. Serum cotinine levels indicate SHS exposure that occurred in the past few days. Nonsmokers exposed to typical levels of SHS usually have serum cotinine concentrations of less than 1 nanogram per milliliter (ng/mL), while active smokers generally have concentrations greater than 15 ng/mL. For this study, participants with serum cotinine concentrations of less than 10 ng/mL were considered nonsmokers (the relatively high cutoff accommodates heavy exposure to SHS).

During the first time period studied, 1988 through 1991, 65% of nonsmokers had serum cotinine concentrations greater than 0.1 ng/mL. On that basis, Healthy People 2010 (a DHHS initiative) had earlier established an objective of reducing that percentage to 45% by the year 2010. The current study results suggest that this goal had already been met by 2000. The authors say the decrease is most likely due to restrictions on smoking at work and in other public places, since adult smoking itself did not decrease dramatically in the 1990s.

Although public health efforts have been successful overall, the results suggest that further efforts should focus on two groups that showed relatively higher levels of risk of SHS exposure—children and

blacks. In the most recent time period studied, 2001 through 2002, the median exposure level for children aged 4 to 11 was almost twice that of adults: 0.067 ng/mL, compared to 0.035 ng/mL. The median exposure level for blacks was even higher, at 0.135 ng/mL, compared to 0.034 ng/mL for whites.

Previous studies have shown that blacks have consistently higher serum cotinine concentrations per cigarette smoked than do whites. But other studies have found higher SHS levels among blacks even after accounting for differences in metabolism. The consistently higher serum cotinine concentrations for black nonsmokers in the current study appear to reflect higher SHS exposure, the authors state, though metabolism differences may have influenced the numbers somewhat.

To focus on these at-risk groups, further public health efforts are needed to discourage smoking where children are present, inside homes, and inside cars, the authors conclude. —**Angela Spivey**



**Signs of progress.** A new study shows that antismoking campaigns have greatly reduced exposures to SHS, but additional measures are still needed to protect at-risk groups such as children and blacks.

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